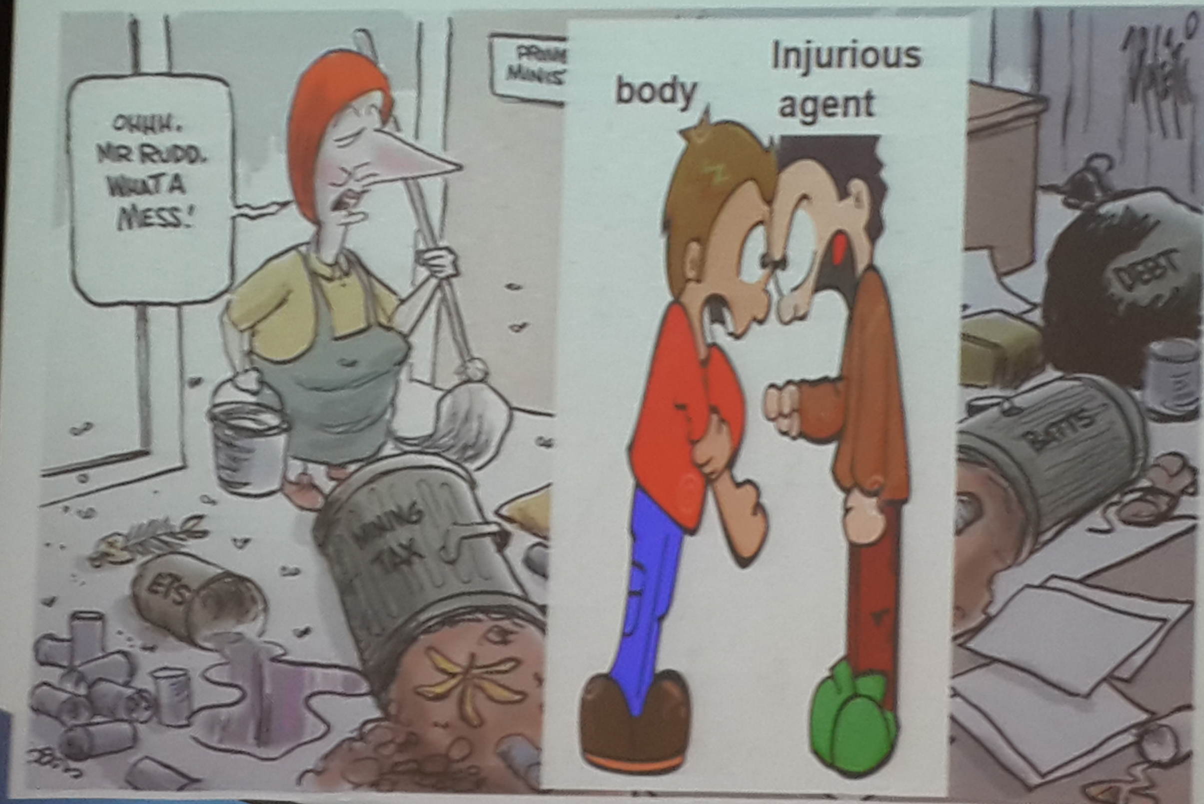
Dr. Riham Abu-Zeid

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Inflammation

Inflammation



Inflammation

Definition:

- It is a protective response of living tissues to eliminate
  - the cause of cell injury
  - the necrotic cells and tissues resulting from that injury.
- This response helps in
  - diluting
  - destroying
  - neutralizing harmful agents e.g. micro- organisms and toxins.

Types of inflammation:

	Acute inflammation	Chronic inflammation
onset	Rapid	Gradual
duration	Short (few minutes up to few days) IF severe →fulminant acute inflammation	Longer duration (days to years) N.B • Chronic active inflammation

5

Causes of acute inflammation

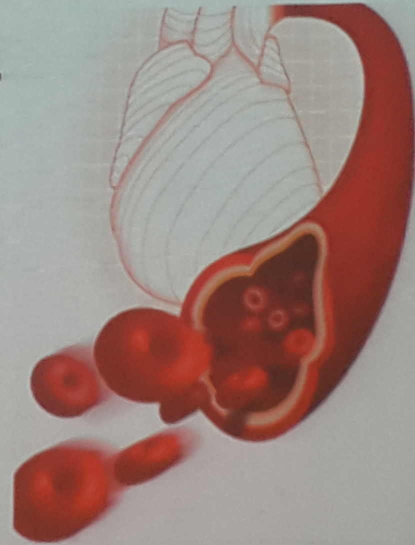
- Infections
- Immune reactions
- Physical agents
- Chemical agents
- Inert materials
- Tissue necrosis

5

1-Acute inflammation

Acute inflammatory response can be divided into two components:

1. **Vascular changes.**



2. **Cellular events.**

1-Vascular changes

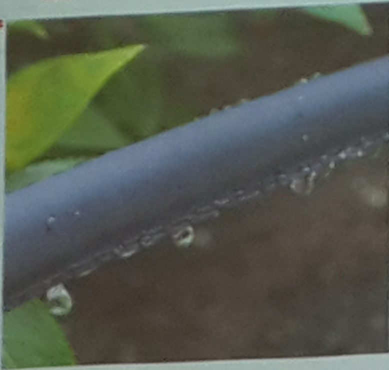
A) Changes in vascular caliber & blood flow

1- Immediate Transient V.C of arterioles

2-Persistent progressive V.D >>>??

B-Increased vascular permeability

2-Persistent progressive vasodilatation

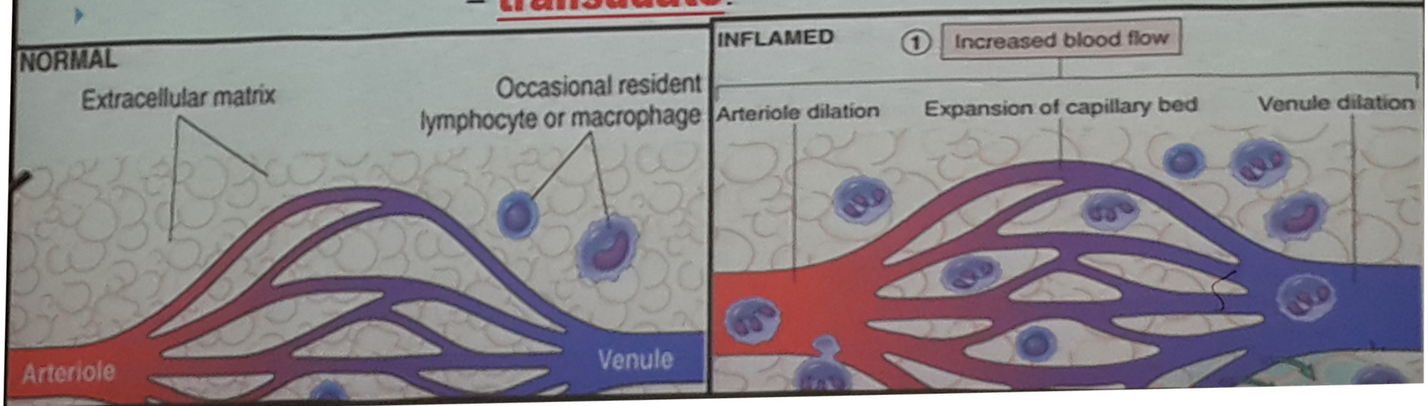




Hyperemia

transudate

- mainly in the **arterioles** within **half an hour** of injury
- ***leads to*** ↑ **blood flow &** ↑**local intravascular hydrostatic pr.** with movement of fluid (plasma containing **little protein**)
  = **transudate**.



B-Increased vascular permeability

Allows the movement of protein-rich fluid and cells (called exudate) into the interstitium



Edema d.t outflow of water & ions into extravascular tissues.

Stasis d.t ↑blood viscosity

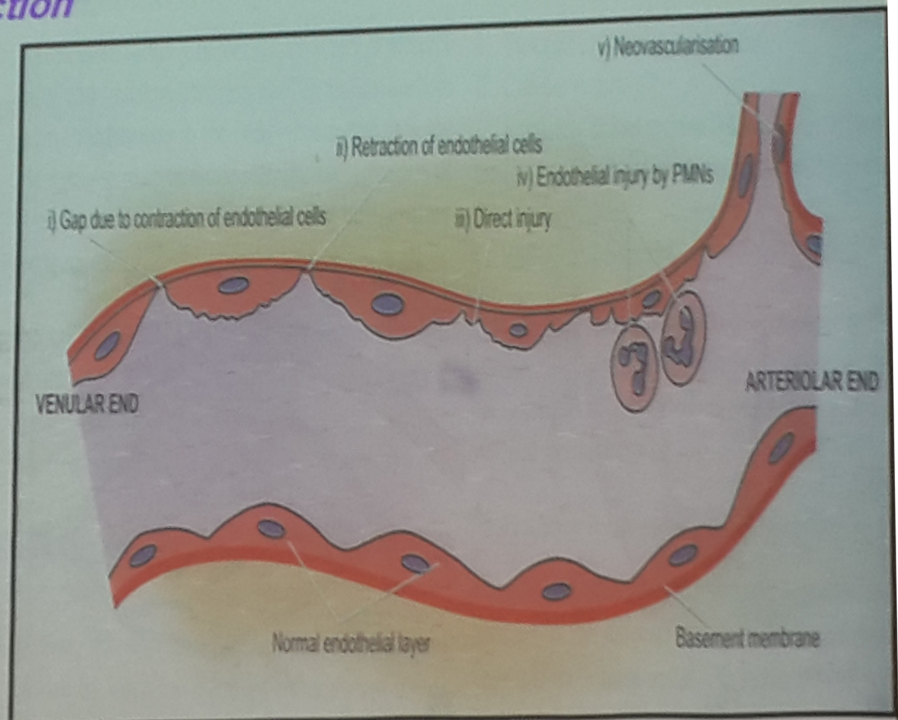
Margination of leucocytes (principally neutrophils) along the vascular endothelial surface

Mechanisms of increased vascular permeability

1-Endothelial cell contraction
Mediated by histamine, bradykinin, leukotrienes

2-Endothelial cell retraction
Mediated by cytokines as TNF & interleukin-1 (IL-1).

3-Endothelial injury



What is cause of edema in inflammation ?

Early dt inc vasodilatation >inc .hydrostatic pressure

Late dt inc. Vasc. permeability

transudate

exudate

2- Cellular events (Leucocyte recruitment & activation)

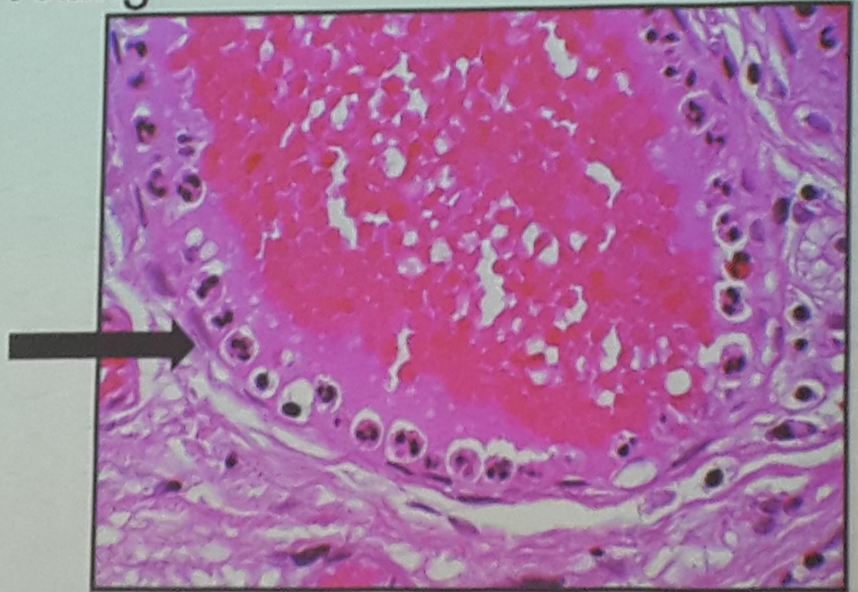
1- Leucocyte Recruitment:

A)Margination and rolling

B)Firm adhesion

C)Transmigration

D)Chemotaxis



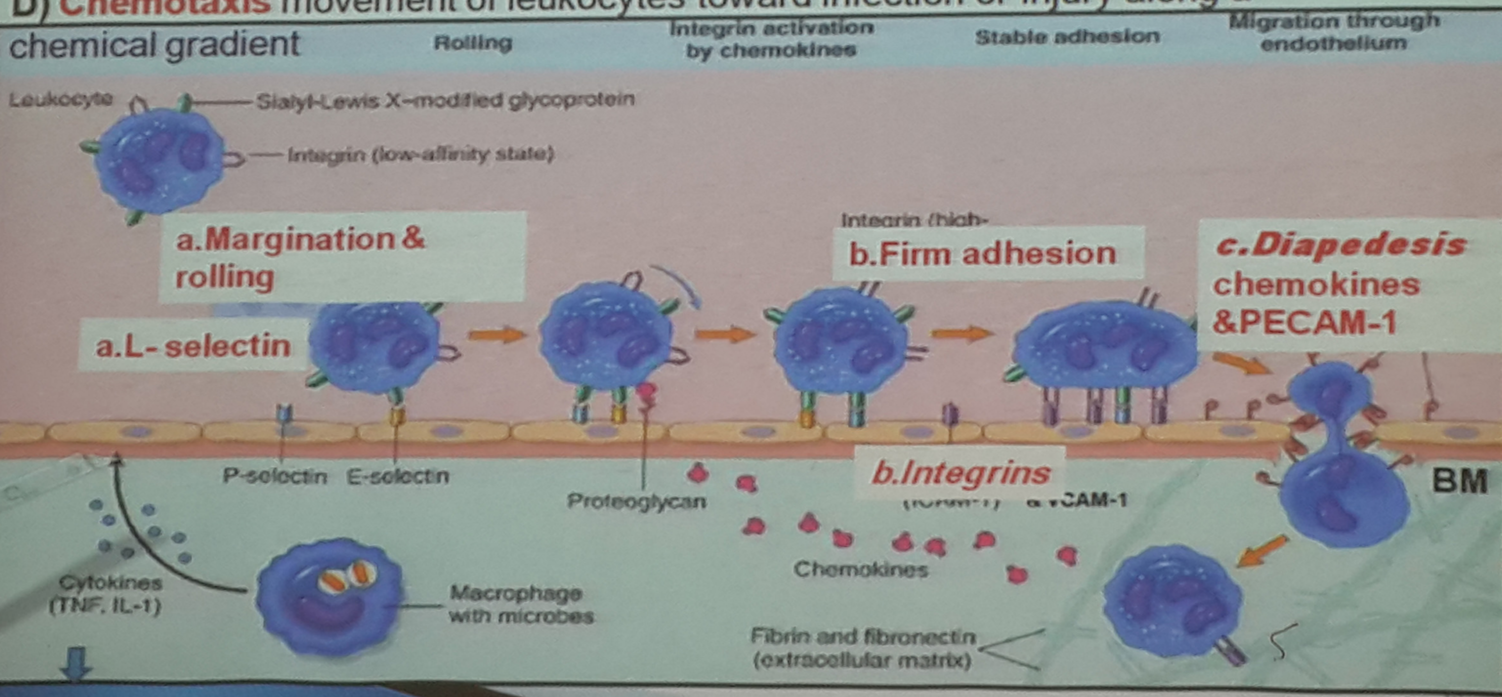
A) Margination and rolling mediated by *selectin family*

B) Firm adhesion mediated by *integrins*

C) Transmigration *through vessel wall by diapedesis* mediated by chemokines &PECAM-1

degrading BM by collagenases

D) Chemotaxis movement of leukocytes toward infection or injury along a chemical gradient



Chemotactic substances

Exogenous and endogenous (from cells or from liver)as:

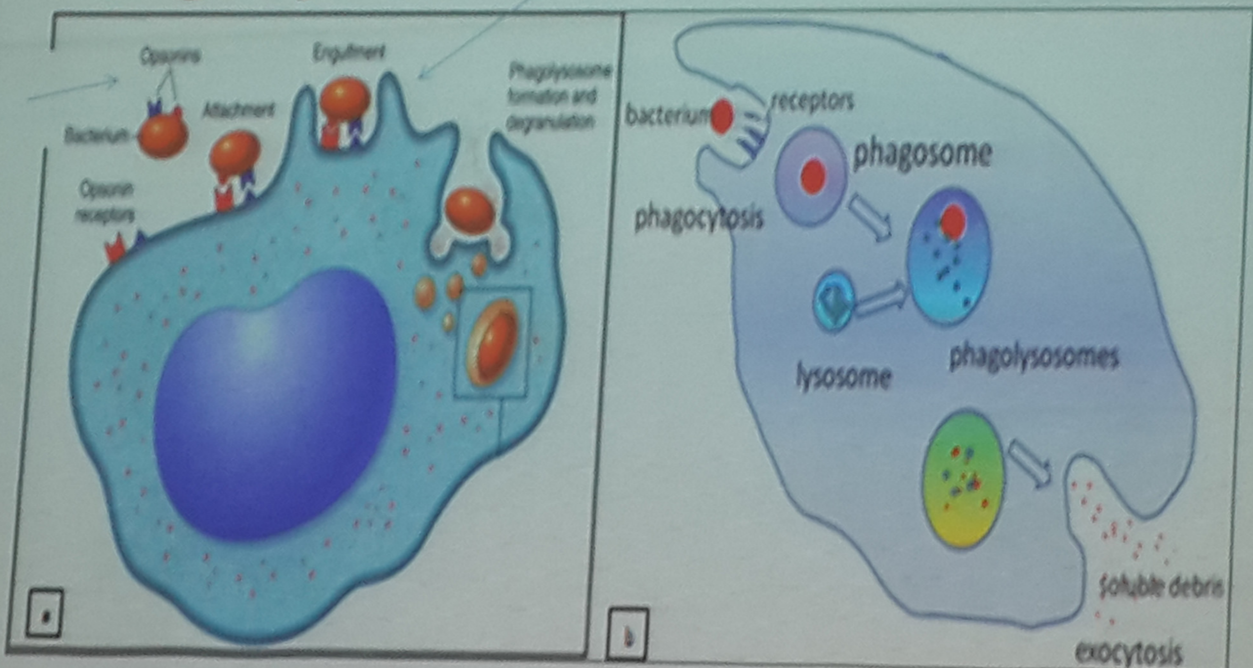
- **Soluble bacterial products.**
- **Cytokines, (IL-8). (Produced by macrophages and other cells)**
- **Components of the complement system, (particularly C5a and C3a).**
- Products of arachidonic acid (AA) metabolism, (leukotriene B_4"LT-B_4").

Chemotactic molecules bind to specific cell surface(inflammatory cells) receptors.

Neutrophils & macrophages ingest bacteria & foreign particles.

Phagocytosis

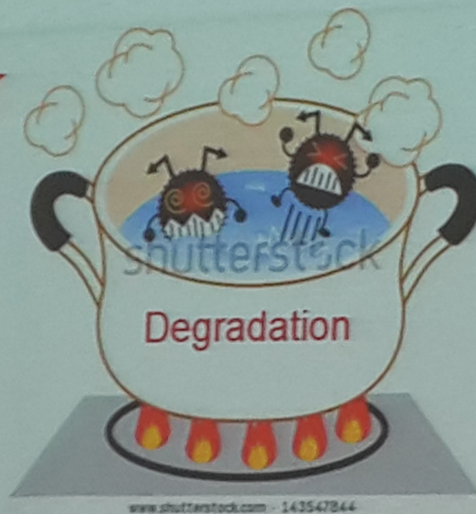
pseudopods



1-Recognition and attachment IgG &C3b=Opsonization
2-Engulfment
3-Killing and Degradation of Micro-organisms

Degradation

A.O2 burst: ROS



B.Lysosomal enzymes of neutrophils

▪NOS

Inflammatory exudate

Pathogenesis

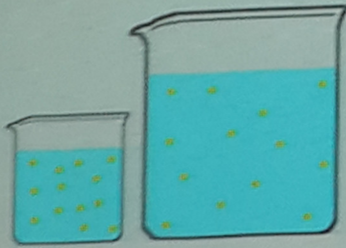
- Increased vascular permeability
- Arteriolar V.D
- Increased osmotic pressure in interstitial fluid d.t splitting of large protein molecules into smaller ones

Composition

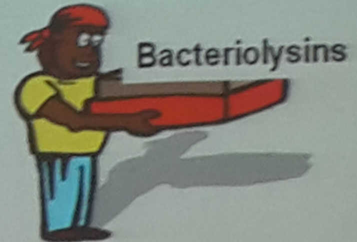
- Plasma or serum rich in fibrinogen
- Neutrophils
- Macrophages (tissue &blood)

Functions

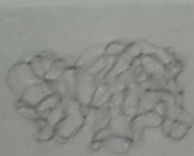
- Dilutes bacterial toxins



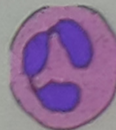
Brings antibodies to area of inflammation
Bacteriolysins→ destroy bacteria
Agglutinins →fix bacteria
Opsonins →coat bacteria to help phagocytosis

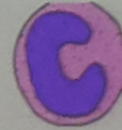


Contains fibrinogens changes to *insoluble fibrin*
network on which leucocyes moves in direction of organisms
localizes infection



- Contains leucocytes
- kill the organisms







neutrophil - produced in bone marrow | monocyte - produced in bone marrow and spleen | lymphocyte - produced in lymph nodes, spleen, and thymus

phagocytes

Test Yourself



One of the following is used by the neutrophils to degrade bacteria

a) Selectins
b) Reactive oxygen species
c) Integrins
d) Prosataglandins

Role of Mediators in Different Reactions of Inflammation

Vasodilatation	Histamine, prostaglandins, nitric oxide
Increased vascular permeability	Histamine and serotonin, Bradykinin substance P Leukotrienes C_4, D_4, E_4
Leucocyte recruitment and Activation	C3a, C5a, IL-1 Bacterial products, Leukotriene B_4.
Fever	IL-1,prostaglandins, TNF
Pain	Prostaglandins, bradykinin
Bacterial degradation & Tissue damage	Lysosomal enzymes of leucocytes Reactive oxygen species Nitric oxide

Local signs in acute inflammation:

1-Redness &Hotness>Why ?

2-Swelling>Why ?

3-Pain>Why ?

- irritation of nerve endings
- PG & bradykinin

4-Loss of function



Morphological patterns of acute inflammation

A-Non suppurative inflammation

- Serous
- fibrinous
- Catarrhal
- Allergic
- Pseudomembranous
- Necrotizing
- Hgic

B-Suppurative inflammation



A-Non suppurative inflammation

1-Serous inflammation=watery fluid





Skin blister

2-Fibrinous inflammation

more severe injury →

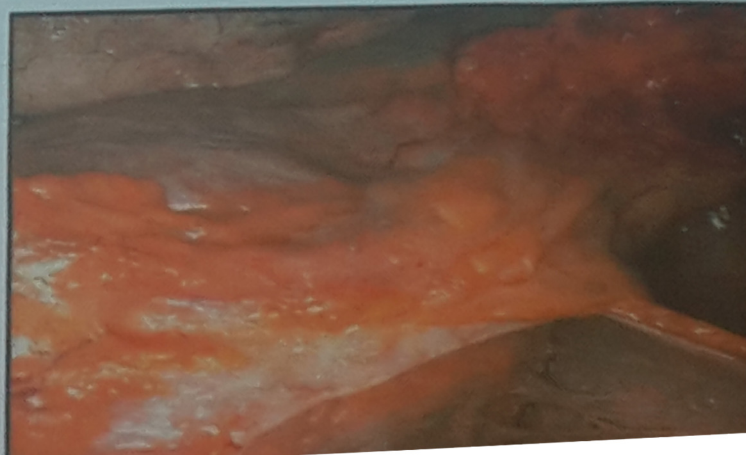
greater vascular permeability

Fibrin rich

e.g.:

Serous sacs.

Lung alveoli in lobar pneumonia.



3- Catarrhal inflammation:

Mild inflammation of m.m

Examples Common cold

Grossly

- **Early** :m.m is red hot swollen dry
- Then there is excess watery mucoid discharge that becomes thick yellow

Microscopically

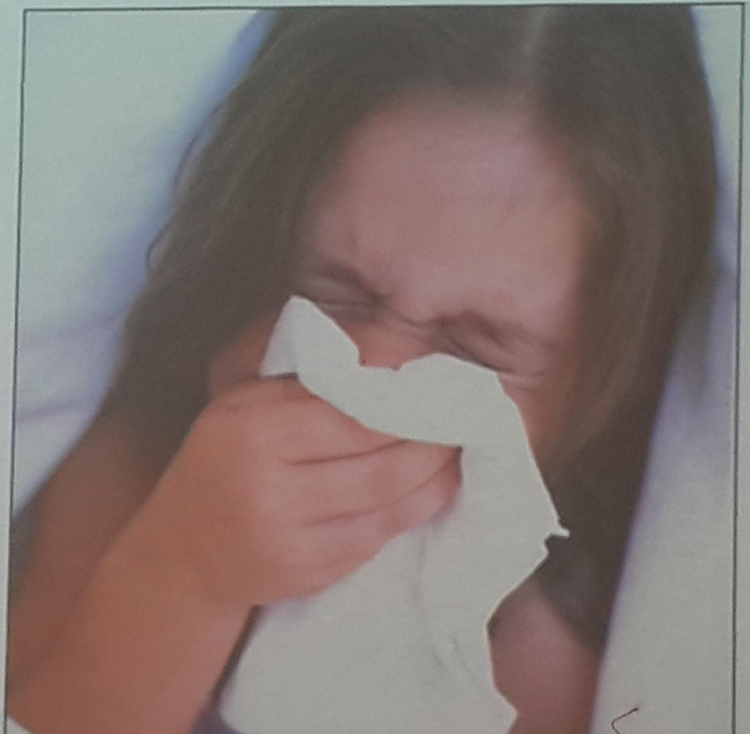
- The mucosal epithelial cells



swollen

d.t to **mucus** accumulation.

- The underlying tissue shows
  - hyperemia,
  - mild edema
  - neutrophil infiltrate.



4-Pseudo membranous inflammation:

- severe inflammation of m.m

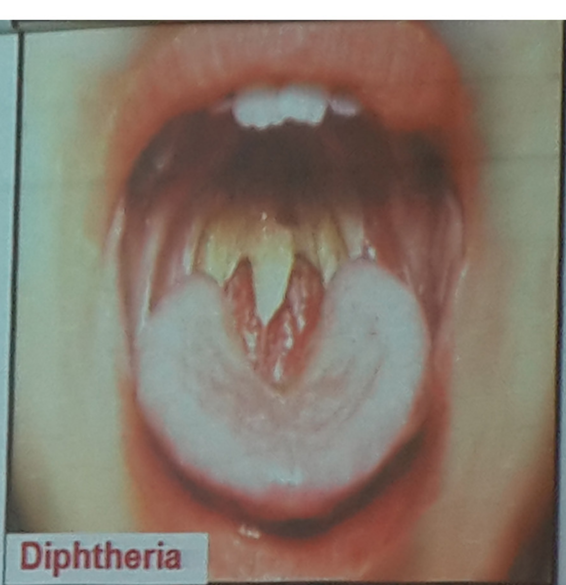
Pathogenesis

- Bacteria → powerful exotoxin → patchy necrosis
- Exotoxin→ pseudomembrane
- **Severe acute toxaemia**

Grossly: pseudomembrane

Mic:

- Causative organism
- Necrotic mucosa
- Fibrin threads
- PNL
- RBCs


Diphtheria



5-Allergic inflammation:

Cause d.t antigen antibody reaction

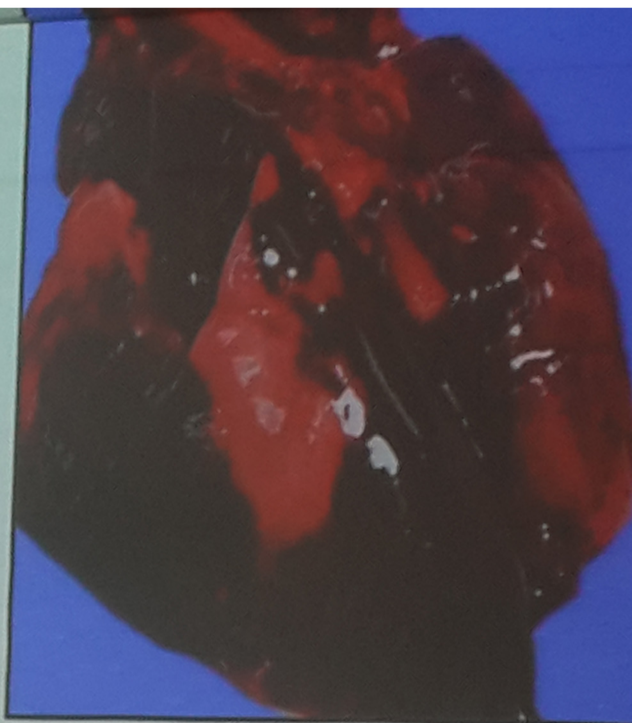
- excess exudates causing edema.
- Increased **eosinophils** in tissue & blood.

Examples

- Urtecaria
- Allergic rhinitis
- Bronchial asthma

6-Haemorrhagic inflammation

- Severe
- destruction of wall of bl. vs with haemorrhage
- as in acute haemorrhagic pneumonia



7-Necrotizing inflammation

- *Inf. with Extensive necrosis*

Q.In acute lobar pneumonia all alveoli are filled with fibrin and inflammatory cells and edema fluid, this type of inflammation is

a.Catarrhal inflammation

b.Fibrinous inflammation

c.Allergic inflammation

d.Pseudomembranous inflammation

e.Suppurative inflammation

A 6-year old girl presented with fever, chills, sore throat with difficulty in breathing. On examination, the whole throat was markedly swollen and red with few areas showing loosely adherent thin grayish white membrane

- What is the possible diagnosis?
- What type of inflammation occurs in this lesion?
- Explain the pathogenesis of this disease

B- Suppurative (purulent) inflammation:

Def

Acute inflammation ccc by pus formation

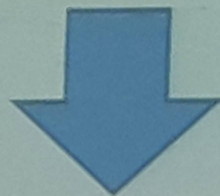
Causative organism

- staph
- strept
- gonoococci
- meningiococci
- E . coli

Pathogenesis of pus formation:

- *Pyogenic organisms→ marked tissue necrosis and strong chemotaxis to PNL*
- many PNL are killed by bacteria → proteolytic enzymes→ liquefaction of necrotic tissue → that mixes with inflammatory exudate→ **pus**

B.Types of suppurative inflammation



-Localized

Abscess

variants

-Furuncle

-Carbancle

-Diffuse

Cellulitis

Abscess

Definition:

Localized suppurative inflammation ccc by cavity containing pus

Cause

- **staph. aureus**
  →<u>coagulase</u> enzyme
  →formation of <u>FIBRIN</u>
  <u>from fibrinogen</u>





to <u>LOCALIZE INFECTION</u>
separate the infected area from the surrounding

Pathogenesis

- The causative organisms are introduced into the tissue causing



marked necrosis & strong chemotaxis to **<u>NEUTROPHILS</u>**



(**central necrotic zone**).

- The vascular phenomena appears at the **peripheral zone** forming



pyogenic membrane

which is composed of many dilated congested capillaries, many neutrophils and the organisms.

Many neutrophils die leading to release of proteolytic enzymes which liquefy the periphery of the necrotic area forming pus

How many zones in the abscess?

3 zones

1. **Central** necrotic zone

2. **Mid zone** containing pus (neutrophils and pus cells)

1. **Peripheral zone** (pyogenic membrane)



N.B:

- The abscess enlarges by further necrosis & liquefaction of the surrounding inflamed zone



until

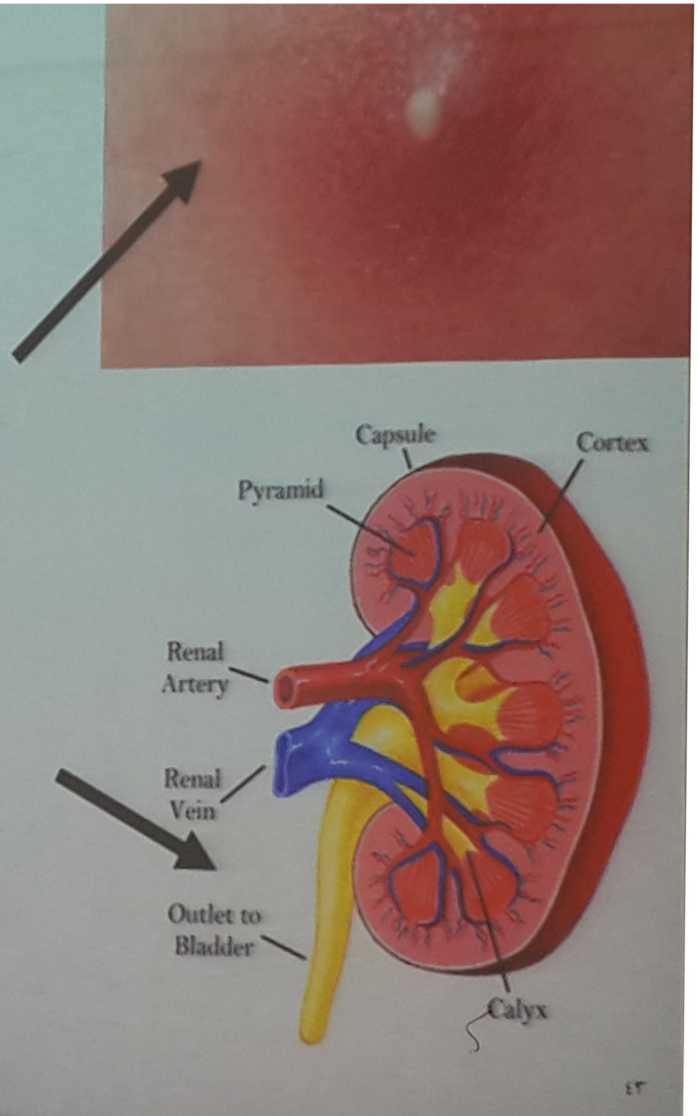
- abscess opens & discharge its contents outside

- The *tension* inside the abscess cavity gradually ↑→pain

Abscess in

* **Sct** →epidermis undergo necrosis and pus escapes

* **kidney** →open into one of the calyces and discharged with urine



Variants of abscess

1-Furuncle (boil)

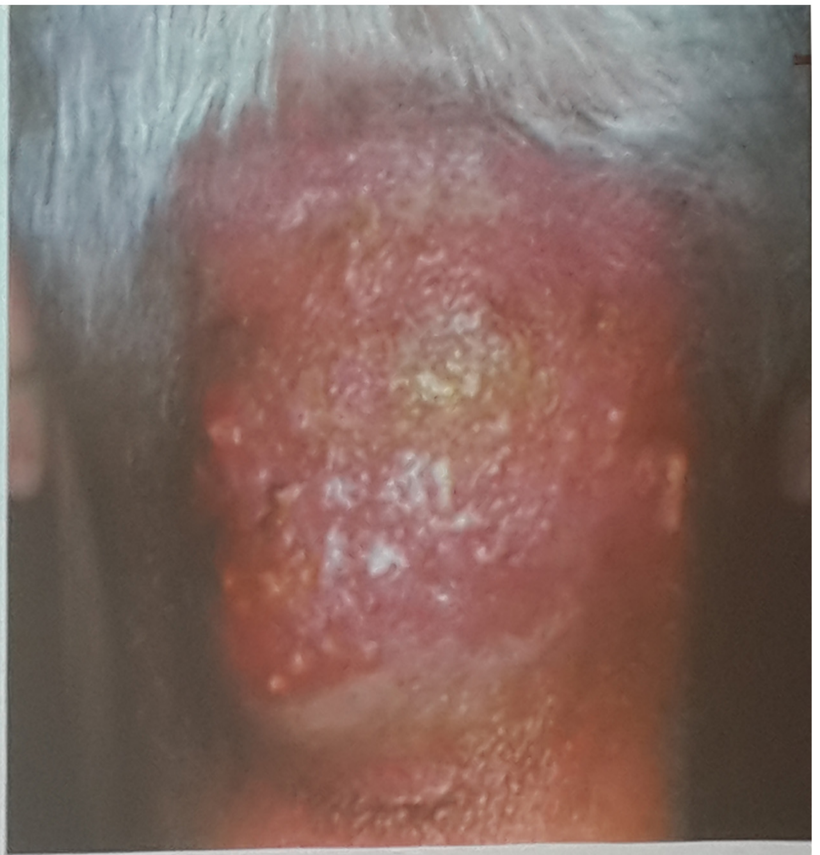
- Small abscess related to **hair follicle or sebaceous gland**
- Caused by Staph. aureus.
- Site: Face, back of neck & axilla





Carbuncle

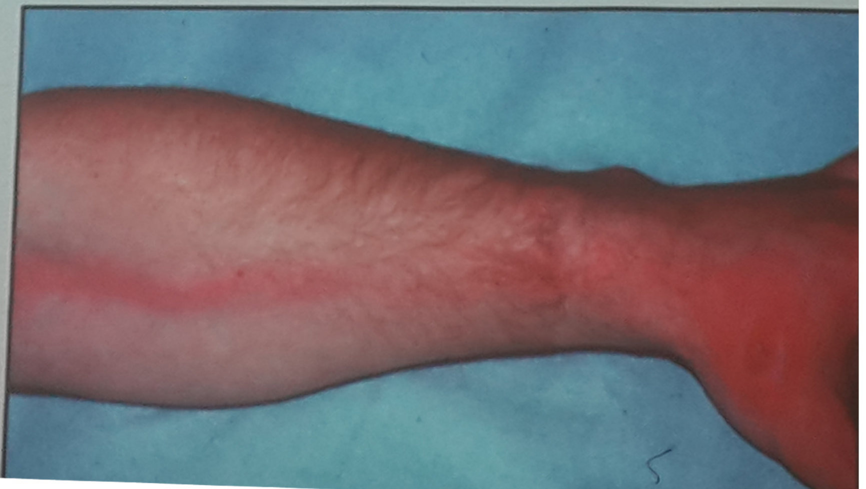
- Pus in multiple loculi separated by f.t strands
- Loculi open on the surface by multiple openings
- Each suppurative loculus develops in the same way as an abscess.
- *Site*: Back of neck & scalp
- Pdf: DM



D.M

Complications of abscess

- Chronic abscess
- Blood spread
- Lymphatic spread
- Complications of healing
  - **Ulcer**
  - **Sinus**
  - **Fistula**



important

Ulcer:

Local defect of skin or mucosal surface d.t necrosis of cells and sloughing or shedding of inflammatory necrotic tissue

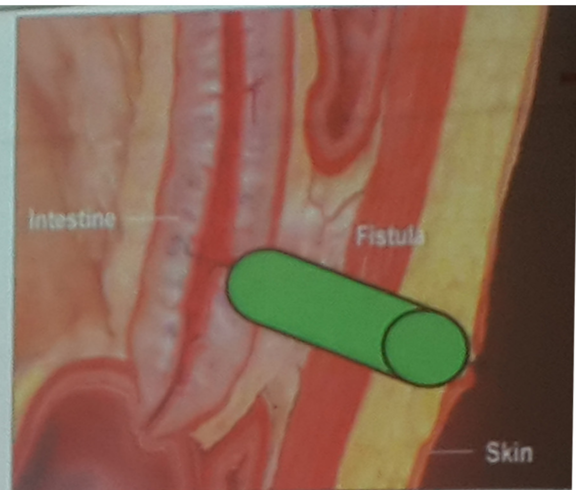


Sinus

- abnormal tract lined by septic granulation tissue
- connecting a cavity to the outside.
- It has a blind end.



Fistula

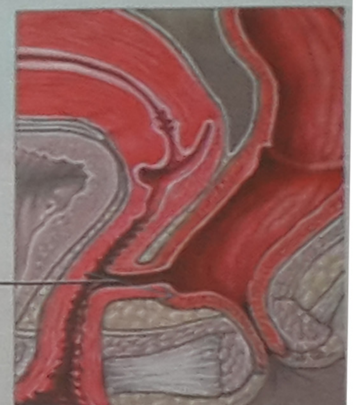


Definition:

- Abnormal tract lined
- by septic granulation tissue

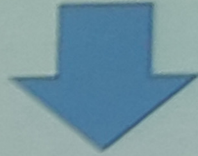
- connecting 2 cavities
- Or between hollow viscera & the surfa

- Differs from sinus as it is opened from both ends.



Vagina & rectum

Types of suppurative inflammation



-Localized

Abscess and its variants

-Diffuse

-Cellulitis

Diffuse suppurative inflammation

Cellulitis

Subcutaneous T.

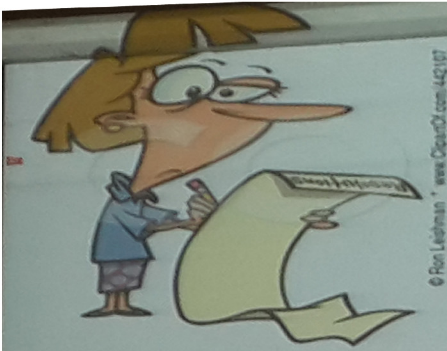


CT of orbit, pelvis or scrotum



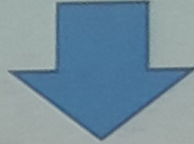
Caustive organism:

- Streptococci →
- streptokinase, fibrinolysin, & hyaluronidase enzymes→
- dissolving matrix →
- spread of infection & prevent its localization



Reminder

B.Types of suppurative inflammation



-Localized

Abscess

-Diffuse

Cellulitis

variants

-Furuncle

-Carbancl

A patient presented with a small circumscribed swelling on his nose red hot and tender with a yellowish area in the center .This is

a.Cellulitis

b.Hemorrhagic non suppurative

inflammation

c.Furuncle (boil)

d.Carbuncle

The causitive organism is mostly........

A 22 year old man presents with sore throat on examination the tonsils are enlarged red and covered by yellowish material

- What is the type of inflammation?
- What are the chemical mediators responsible for the pain?
- Name one organisms that can cause this type of inflammation
- Explain how this yellow material is formed

Thank You

